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Case Report

Traumatic dissection of the anterior cerebral artery secondary to a rugby related impact: A case report with emphasis on the usefulness of T1-VISTA

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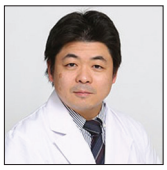

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ABSTRACT

Background: Cerebrovascular injuries (CVIs) are not usually considered in the differential diagnosis of sport-related head injuries (SRHIs). We encountered a rugby player with traumatic dissection of the anterior cerebral artery (ACA) after impact on the forehead. Head magnetic resonance imaging (MRI) with T1-volume isotropic turbo spin-echo acquisition (VISTA) was used to diagnose the patient.

Case Description: The patient was a 21-year-old man. During a rugby tackle, his forehead collided with the forehead of an opponent. He did not have a headache or disturbance of consciousness immediately after the SRHI. On the 2nd day of illness, he had transient weakness of the left lower limb several times. On the 3rd day of illness, he visited our hospital. MRI revealed occlusion of the right ACA and acute infarction of the right medial frontal lobe. T1-VISTA revealed intramural hematoma of the occluded artery. He was diagnosed with acute cerebral infarction due to dissection of the ACA and was followed up for vascular changes with T1-VISTA. The vessel had recanalized and the size of the intramural hematoma had decreased 1 and 3 months after the SRHI, respectively.

Conclusion: Accurate detection of morphological changes in cerebral arteries is important for the diagnosis of intracranial vascular injuries. When paralysis or sensory deficits occur after SRHIs, it is difficult to differentiate between concussion from CVI. Athletes with red-flag symptoms after SRHIs should not merely be suspected to have concussion; they should be considered for imaging studies.

Keywords: Concussion, Rugby, Sport-related head injury, T1-volume isotropic turbo spin-echo acquisition, Traumatic anterior cerebral artery dissection

INTRODUCTION

In recent years, education and awareness regarding the management of sport-related head injuries (SRHIs), especially concussions, have been emphasized and promoted.[8] However, due to lack of a clear evaluation criteria, the diagnosis remains difficult. There is a concern that athletes with so called red-flag symptoms (headache that worsens, seizures, slurred speech, increasing confusion, repeated vomiting, unequal pupil size, very drowsy, weak or numb arms

or legs, unusual behavior, increasing irritability, and loss of consciousness) after an SRHI may have suffered traumatic brain injury.[15] Most types of intracranial hemorrhage such as subdural hematoma, epidural hematoma, and subarachnoid hemorrhage are commonly considered in the differential diagnosis of SRHIs;[22] in contrast, cerebrovascular injuries (CVIs) are rarely contemplated.[4,16] Furthermore, most of the few reports on sport-related CVIs refer to cervical vascular injuries and reports of intracranial vascular injuries are scarce.[1,22]

We present a rugby player who had a right frontal lobe infarction due to traumatic dissection of the anterior cerebral artery (ACA), which resulted in transient left lower extremity paralysis after a hard blow to the forehead. The team medical staff suspected he had a concussion. Patient was quickly evaluated by magnetic resonance imaging (MRI) with T1-volume isotropic turbo spin-echo acquisition (VISTA) (Philips Medical Systems, Eindhoven, Netherlands).

CASE DESCRIPTION

A 21-year-old male college student presented with no previous medical history or any drug intake. During a tackle at rugby practice, his forehead collided with the forehead of an opponent. Immediately after the collision remained asymptomatic and continued to play. However, several hours later of the same day, the patient started experiencing a mild headache with a gnawing pain all over the head. On the 2nd day, he took part in another rugby practice. However, during the rugby practice, experienced several episodes of transient left lower extremity paralysis reporting the event to the team trainer that suspected a concussion and immediately ordered him to stop playing. On the 3rd day of illness, he reported to the team doctor, who instructed him to visit our hospital.

Findings at presentation

The patient did not have headache or posterior neck pain. His Glasgow coma scale score was 4-5-6 and he did not have pupillary abnormalities, nystagmus, eye movement disorder, tetraplegia, sensory disturbance, or balance disorder. The finger-nose test did not reveal any abnormality in the patient. Similarly, his blood tests showed no abnormalities. His electrocardiogram showed sinus rhythm and a pulse rate of 56/min.

Imaging examination

Computed tomography of the head did not show intracranial hemorrhage, but low absorptive changes were observed in the medial right frontal lobe. Magnetic resonance angiography (MRA) showed occlusion of the right ACA. T1-VISTA revealed an intramural hematoma (hyperintense intramural signal) that was consistent with the occluded artery [Figures 1 and 2a]. Carotid artery echocardiography showed no abnormality. Based on the above-mentioned results, the patient was diagnosed with acute cerebral infarction due to traumatic ACA dissection.

Course of events after hospitalization

He was admitted urgently on the 3rd day of illness and underwent thorough neurological examination and blood pressure monitoring. Since the CVI was caused by dissection, there is a possibility that an aneurysm may form in the future that could cause a cerebral hemorrhage. Therefore, the patient was not administered antithrombotic therapy. After admission, the patient was closely monitored for blood pressure, which was not high enough to require antihypertensive medication. He remained clear and conscious with no recurrence of headache or lower extremity paralysis. It was decided to evaluate his vessel with a minimally invasive MRI. If the MRI showed cerebral infarct enlargement or aneurysm formation, a digital subtraction angiography (DSA) was scheduled for a more detailed evaluation. T1-VISTA was repeated on the 10th illness day; it did not show recanalization of the occluded artery and no aneurysm had formed [Figure 2b]. On the 11th day of illness, he was discharged home with a modified Rankin scale score of 0. After discharge, he underwent a follow-up MRI.

T1-VISTA performed 1 month after the SRHI (i.e., on the 31st day of illness) showed recanalization of the occluded artery, but no aneurysmal changes were observed [Figure 2c]. In addition, the intramural hematoma had extended to the periphery of the artery. T1-VISTA performed 3 months after the SRHI (i.e., on the 82nd day of illness) showed maintained recanalization, but it did not show aneurysm formation or any other morphological abnormality of the artery. It also showed that the size of the intramural hematoma had decreased [Figure 2d].

Our plan was to perform DSA if there were any changes in vessel morphology over time and to consider the need for additional treatment. In this case, he was able to be followed the changes in vascular morphology using MRI and T1-VISTA. Based on the above clinical and radiological course, we decided that the patient would continue to be followed conservatively and that detailed examination with DSA was unnecessary.

DISCUSSION

The etiology and incidence rate of sport-related CVI are unclear. It was reported in most previous studies that sport-related CVIs involve extracranial carotid or vertebral artery dissection and may cause cerebral infarction.[1] There is a risk of cerebral infarction in a wide variety of sports that involve

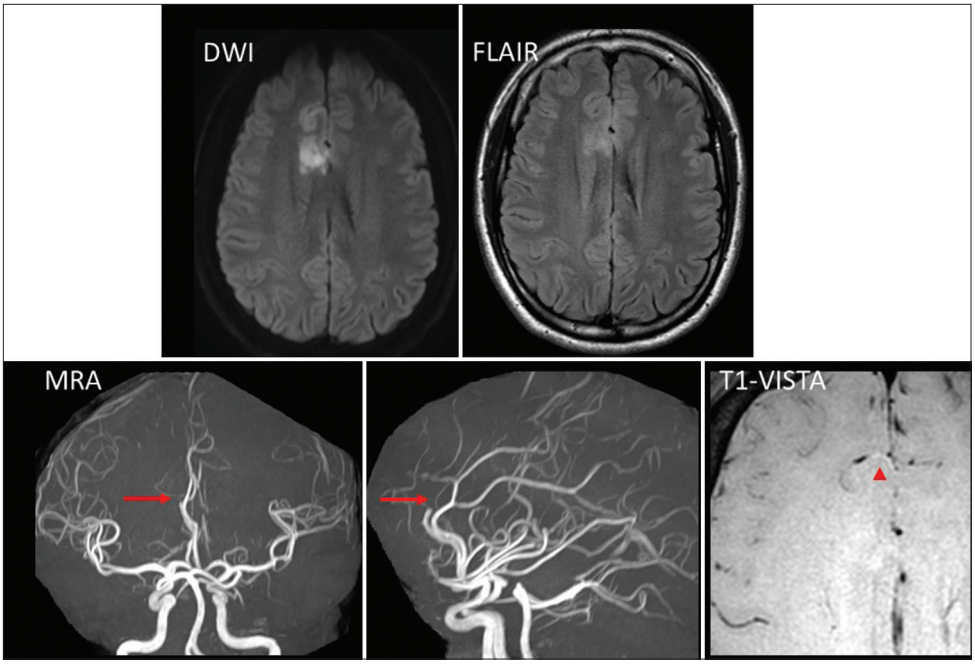

Figure 1: Head magnetic resonance imaging and magnetic resonance angiography (MRA) on admission diffusion-weighted imaging and fluid-attenuated inversion recovery showing acute cerebral infarction in the medial right frontal lobe, MRA showing occlusion of the right anterior cerebral artery (→) and T1-volume isotropic turbo spin-echo acquisition (T1-VISTA) showing an intramural hematoma with high signal intensity consistent with the occluded vessel (▲). Red arrows indicate the occluded right anterior cerebral artery. Red arrowhead indicates intramural hematoma. DWI ; diffusion weighted image, FLAIR ; fluid attenuated inversion recovery.

cervical motion such as contact sports like rugby and non-contact sports such as bowling, yoga, and golf.[3,5,17] Blunt trauma to the neck and rotational impact or carotid artery stretch during extreme neck extension is known causes of common and internal carotid artery dissection.[3] In the case of rugby, CVIs primarily due to carotid artery dissection caused by blunt trauma to the neck have been reported.

Dissection of intracranial cerebral arteries can result in cerebral infarction due to vascular occlusion or intracranial hemorrhage due to aneurysm formation. Intracranial cerebral aneurysms caused by traumatic dissection are rare, accounting for <1% of all intracranial aneurysms. It is estimated that 30% of all intracranial cerebral aneurysms involve the ACA.[7,24] Aggressive surgery should be planned to treat aneurysms caused by dissection because these aneurysms are associated with a high risk of rupture. Further, occlusion of the ACA due to traumatic intracranial artery dissection is uncommon. The incidence of cerebral infarction due to intracranial artery dissection after severe head trauma is 1.9–11.9%, depending on the severity of the trauma; it is even higher in recent years, as demonstrated by the increasing number of case reports.[2,19] However, reports of dissection after SRHI are rare and the occurrence of dissection after SRHI is not easily detectable.[6]

The ACA is thought to be injured at the limb of the falx cerebri by migration of brain tissue or by traction forces due to its anchorage to the falx cerebri.[23,25] Traumatic changes in arteries include damage to the tunica intima (or the inner layer) of arteries, resulting in hemorrhage within the arterial wall, dissection, and false lumen formation. Subintimal dissection between the tunica intima and tunica media results in narrowing or occlusion of the arterial lumen due to inward swelling, while subintimal dissection between the tunica adventitia and tunica media results in outward swelling and aneurysm formation.[26] Thus, accurate detection of morphological changes in cerebral arteries is important for the diagnosis of intracranial vascular injuries.[10]

Intracranial vascular injuries are often caused by high-energy head trauma, such as car accidents and falls.[14] In our case, the forehead was directly impacted during a rugby tackle. It has been reported that a rugby scrum could cause an impact of 4.4 kN in a male high school player and 8.0 kN in a male Australian national team player.[16] Given that the force required to cause skull fracture is estimated to be 3.5–5.8 kN,[11] it is quite possible that high-energy impact during contact play in rugby (depending on the level of competition) could result in intracranial damage.

Morphology is important in the diagnosis of dissection. Failure to accurately assess the morphology of dissection

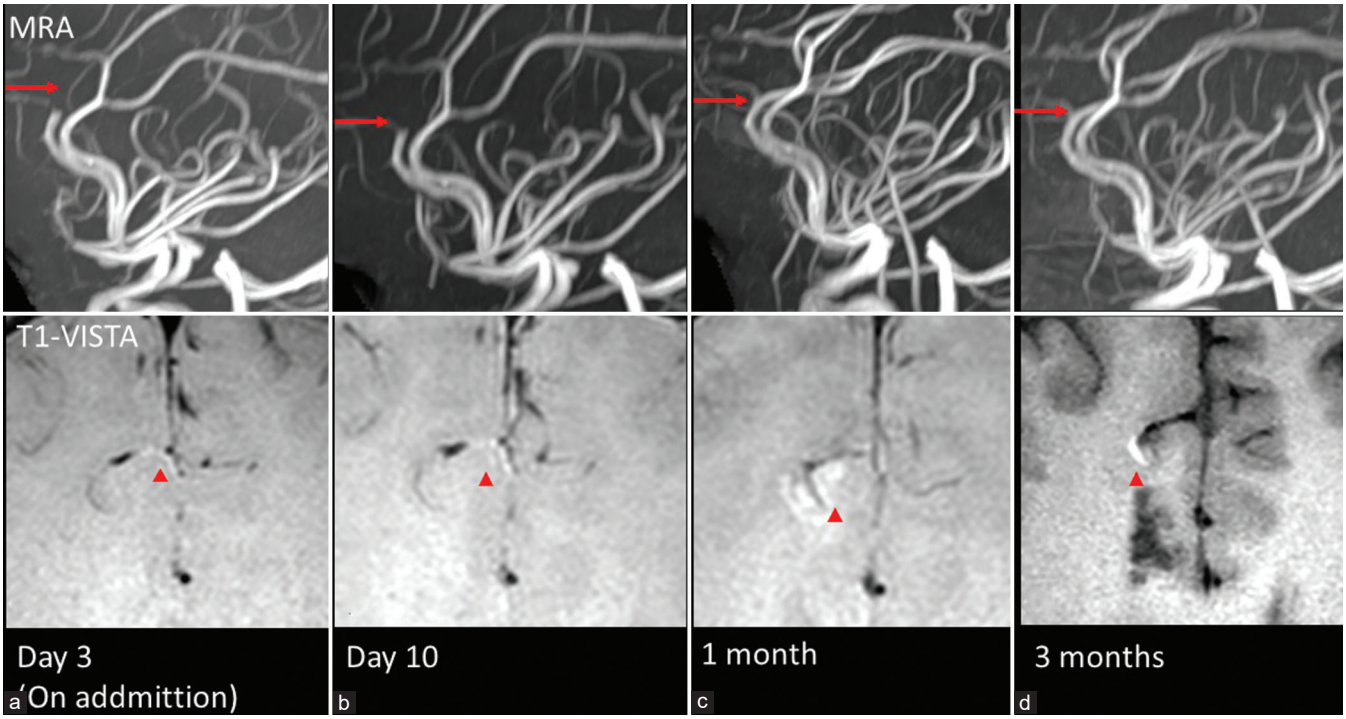

Figure 2: Follow-up of changes in head Magnetic resonance angiography (MRA) and T1-volume isotropic turbo spin-echo acquisition (T1-VISTA) We monitored the occluded artery (→) and the intramural hematoma (▲) with MRA and T1-VISTA. Red arrows in all figures indicate the occluded artery. Red arrowheads in all figures indicate the intramural hematoma. T1-VISTA was repeated on the 10th day of illness, 1 month after the sport-related head injury (SRHI) and 3 months after the SRHI. (a) MRA and T1-VISTA on the 10th day of illness showing no vascular recanalization and no aneurysm formation. (b) Recanalization of occluded artery with no aneurysmal changes observed 1 month after the collision. (c) Reocclusion, aneurysm formation or any other morphological abnormality of the artery not observed 3 months after the SRHI. The intramural hematoma extended to the peripheral artery over time, but by 3 months after the SRHI, the size of the intramural hematoma had decreased (d).

leads to delay in diagnosis. Using MRA and T1-VISTA, we were able to diagnose occlusion of a peripheral branch of the ACA that was caused by dissection. It was reported in recent studies that three-dimensional high-resolution vessel wall imaging facilitated the evaluation of intracranial arteries.[12,27] Another study reported that VISTA has less magnetic susceptibility to artifacts and can collect a wider range of images than time-of-flight MRA.[21] VISTA is useful in the diagnosis of cerebral artery dissection because of its short acquisition time and non-invasiveness. In addition to morphological changes in vessels, detection of specific features such as intimal hematoma is important in the diagnosis of dissection. T1-VISTA can be used for the three-dimensional evaluation of intravascular plaques and intimal hematoma.[12] Although cerebral angiographic findings such as double lumen, intimal flap, pearl-and-string sign, and string sign are considered gold standards for the imaging diagnosis of intracranial artery dissection, it is difficult to accurately assess the shape changes of peripheral arteries. Intramural hematoma, as in our patient, is considered a definitive finding of dissection in peripheral arteries and a hematoma in a false lumen that can be visualized by T1-VISTA as a hyperintense intramural signal; it may be a useful finding in the diagnosis of intracranial arterial dissection.[9,13]

In this case, T1-VISTA was used to evaluate the morphological changes in the artery and the intramural hematoma over time. Intracranial cerebral artery dissection may worsen within 1–2 weeks of onset, with progressive stenosis and enlargement of the aneurysm, but thereafter, the stenosis and occlusion often improve and the aneurysm shrinks. Therefore, it is advisable to follow-up on patients for accurate detection of vascular changes within at least 3 weeks of onset.[18]

Although the importance of responding to concussions is becoming more widely disseminated, the diagnosis of concussion is not always straightforward. Not all athletes with suspected concussion require hospital evaluation and imaging studies may not be performed if an athlete is deemed only mildly ill after a hospital visit.[20] Concussions can present a wide variety of symptoms. Since the transient motor and sensory deficits experienced after concussions overlap with symptoms of stroke, it is sometimes difficult to differentiate between cerebral infarction and concussion;

thus, imaging studies may play an important role in diagnosis. Athletes suspected to have concussion after an SRHI should be considered for imaging studies, especially if they have red-flag symptoms. It is sometimes risky to diagnose concussion simply because an athlete had an SRHI. Furthermore, athletes with suspected concussion, especially those with red-flag symptoms, should be considered for imaging studies to evaluate them for other traumas, such as CVIs.

CONCLUSION

We presented the case of a rugby player who had frontal infarction due to traumatic ACA dissection. When evaluating SRHIs, CVIs should be included in the differential diagnosis and it is important to evaluate neck arteries as well as intracranial arteries. T1-VISTA is useful in the diagnosis of cerebral artery dissection, and it can be used to monitor vascular changes over time.

Declaration of patient consent

Institutional Review Board (IRB) permission obtained for the study.

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Conflicts of interest

There are no conflicts of interest.

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